
**Reply**

We appreciate the commentary provided by Drs. Volders and Daemen regarding our work on postinfarct ventricular remodeling (1).

The crux of their critique relates to the fact that our observations deny the widely accepted concept that postinfarct myocardial hypertrophy, as an adaptive phenomenon to the loss of working myocardium, is associated with interstitial fibrosis per se. This concept has led to the inevitable conclusion that the clinical phenomenon of progressive heart failure is because of the increase in extracellular matrix components. Our observations reveal that this sequence of events is not necessarily true. Indeed, the patients reported in our study all presented clinically with progressive heart failure, which necessitated heart transplantation as the only remedy and, interestingly, neither one of these individuals had a significant increase in interstitial fibrous tissue in the noninfarcted hypertrophied myocardium. Hence, the message is that postinfarct remodeling in our patients is not associated with interstitial fibrosis, despite progressive clinical deterioration.

It appears as if Drs. Volder and Daemen wish to ignore the fact that the clinical condition deteriorated despite absence of interstitial fibrosis in the noninfarcted myocardium. Instead, they focus on the potential role of angiotensin-converting enzyme (ACE) inhibitors to “explain” the lack of fibrosis, an aspect that we dealt with in the paragraph “Study limitations.” It is of interest that otherwise Drs. Volders and Daemen provide no data to contradict our observations. Thus, we are left with the impression that Drs. Volders and Daemen are biased by their own studies on this subject. Be that as it may, if the use of ACE inhibitors in our patients does account for the lack of interstitial fibrosis in the noninfarcted myocardium, the search for tools to prevent an increase in extracellular matrix and, hence, clinical deterioration is not necessarily the solution in patients with postinfarct myocardial remodeling. This is precisely the point made in our final paragraph.

ANTON E. BECKER, MD, FACC
Department of Cardiovascular Pathology
Academic Medical Center
Meibergdreef 9
1105 AZ Amsterdam, The Netherlands
E-mail: m.i.schenker@amc.uva.nl

MONIQUE M.M.H. MARIJANOWSKI, PhD
Department of Medicine
Emory University School of Medicine
1364 Clifton Road NE
Atlanta, Georgia 30322

Table 1: Incidence of Cardiovascular Death in the Tsuna Region after the 1995 Hanshin–Awaji Earthquake

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>January</th>
<th>February</th>
<th>March</th>
<th>April</th>
<th>Total (Jan–Apr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>13 (9)</td>
<td>15 (4)</td>
<td>10 (14)</td>
<td>7 (4)</td>
<td>45 (31)</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>11 (0)</td>
<td>4 (3)</td>
<td>8 (2)</td>
<td>5 (1)</td>
<td>26 (6)</td>
</tr>
<tr>
<td>Sudden death</td>
<td>2 (9)</td>
<td>11 (1)</td>
<td>2 (12)</td>
<td>2 (3)</td>
<td>17 (25)</td>
</tr>
<tr>
<td>Stroke</td>
<td>9 (3)</td>
<td>25 (10)</td>
<td>15 (7)</td>
<td>9 (11)</td>
<td>59 (31)</td>
</tr>
<tr>
<td>Cerebral infarction</td>
<td>7 (2)</td>
<td>19 (5)</td>
<td>8 (2)</td>
<td>7 (6)</td>
<td>41 (15)</td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
<td>2 (1)</td>
<td>3 (2)</td>
<td>5 (2)</td>
<td>1 (3)</td>
<td>11 (8)</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>0 (0)</td>
<td>1 (1)</td>
<td>2 (1)</td>
<td>1 (2)</td>
<td>4 (4)</td>
</tr>
<tr>
<td>Unclassified stroke</td>
<td>0 (0)</td>
<td>2 (2)</td>
<td>0 (2)</td>
<td>0 (0)</td>
<td>2 (4)</td>
</tr>
<tr>
<td>All cardiovascular disease</td>
<td>22 (12)</td>
<td>40 (14)</td>
<td>25 (21)</td>
<td>16 (15)</td>
<td>103 (62)</td>
</tr>
</tbody>
</table>

(1) to conclude that there is no increase in stroke in whites after the major quake, a longer period of study after the quake is necessary.

KAZUOMI KARIO, MD, PhD
Department of Cardiology
Jichi Medical School
Yakushiji, Minamikawachi
Kawachi, Tochigi, 329-04, Japan
E-mail: kkario@jichi.ac.jp

References

Reply
We thank Dr. Kario for his interest in our recent article (1). Both his study (2) and ours showed an increase in cardiac death rate associated with a major morning-time earthquake. In our study the increased event rate was confined to the day of the earthquake and then fell, whereas in his study there were continued cardiac events as well as cerebral vascular events for several months thereafter. There were clear differences in the types of analyses performed, locations of the studies, possibly ages of the patients and racial differences of the populations, and in general we agree with Dr. Kario that these might help explain the differences in duration during which cardiac events occurred. There are some other possible explanations. It is our understanding that the degree of direct physical trauma and destruction of housing was overall more concentrated and severe in the Hanshin–Awaji earthquake, which may have resulted in continued psychological stress for the victims and their families. This increased and continued stress may have contributed to continued cardiac events. Other factors such as the magnitude, severity and timing of aftershocks also may have played a role. Finally, we analyzed cardiac deaths on a day-by-day basis rather than by month. The number of cardiac deaths we reported in Los Angeles County (population of about 9 million) were significantly greater day to day than those in Dr. Kario’s report.

We think it is important to study the effects of various stressors on cardiac events, and clearly all natural disasters do not have the same effects on outcome. Thus although both the Northridge earthquake and Hanshin–Awaji earthquakes were associated with increased cardiac events, these were reduced in the weeks that followed the Northridge earthquake whereas they continued in the weeks and months after the Hanshin–Awaji earthquake. It is interesting that both of these earthquakes that were associated with an initial increase in cardiac events occurred in the early morning hours (3). Waking up to such a quake is frightening (personal experience of the author who lives ~10 miles from the epicenter of the Northridge quake) and certainly the wake-up time has been found to be a trigger of various cardiac events. Waking up to a natural disaster most likely compounds this problem. Of note, the 1989 Loma Prieta quake, which occurred in the late afternoon (5:04 PM) in Northern California was not associated with an increase in cardiac events (4).

ROBERT A. KلونER, MD, PhD
JONATHAN LEOR, MD
The Heart Institute
Good Samaritan Hospital
1225 Wilshire Boulevard
Los Angeles, California 90017–2395

References